LIPID PROFILE CHANGES IN PREGNANCY INDUCED HYPERTENSION

- A CASE CONTROL STUDY

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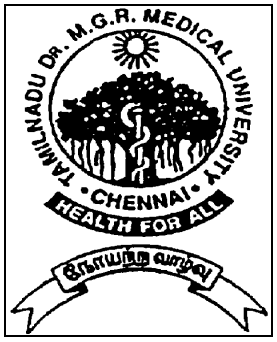

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CERTIFICATE

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INDEX

Introduction

INTRODUCTION

Preeclampsia is a leading cause of maternal and perinatal morbidity and mortality world wide. The disorder is commonly defined by hypertension and proteinuria arising in the second half of pregnancy in a previously normotensive woman.

PATHOLOGY

In preeclampsia, there is a shallow invasion of the spiral arteries by the trophoblast resulting in inadequate placentation. Furthermore, the placenta contains more fibrin deposits and thrombosis as compared to uncomplicated pregnancy. This results in a poorly perfused fetoplacental unit. The subsequent placental hypoxia may amplify the release of inflammatory stimuli in the maternal circulation, during dysfunctional endothelial cell activation.

Endothelial cell dysfunction is a key feature of the pathogenesis of preeclampsia. In preeclampsia, characteristic pathological lesions in the placenta are fibrin deposits, acute atherosis and thrombosis. The similarity between the lesions of preeclampsia and atherosclerosis has led to speculations of a common pathophysiological pathway. An abnormal lipid profile is known to be strongly associated with atherosclerotic cardiovascular disease and has a direct effect on endothelial cell activation. Abnormal lipid metabolism seems to be important in the pathogenesis of preeclampsia too.

Normal human pregnancy results in a pronounced physiologic

hypertriglyceridemia involving a gestational rise in blood triglycerides and cholesterol. Serum triglycerides and LDL concentrations in women with preeclampsia were higher than those in women with uncomplicated pregnancy.

During the first half of normal pregnancy, increased maternal fat accumulation (relative anabolic state) is presumed to be important for the subsequent hypertriglyceridemia normally occurring in later gestation (relative catabolic state). Circulatory concentrations of VLDL & LDL normally increase with gestational age as reflected by marked increases in serum triglycerides and cholesterol. The hypertriglyceridemia is due primarily to enhanced entry of triglyceride rich lipoproteins (esp VLDL) into the circulation rather than to diminished removal. Estrogen may play a major role in the lipoprotein patterns seen in human pregnancy although LDL cholesterol is more influenced by the combined effect of increased estrogen and progesterone. Additionally placental lipoprotein lipase activity normally increases as term approaches.

The mechanisms driving the abnormal elevation in triglycerides and free fatty acids in preeclampsia are unclear metabolic patterns resembling `Syndrome X' or `Insulin Resistance Syndrome'. Heightened insulin resistance occurring in preeclampsia would increase fatty acid mobilisation from the visceral fat, promote overproduction of VLDL by the liver and suppress activity of posthepatic lipoprotein lipase resulting in elevated serum free fatty acids and triglycerides.

Review of Literature

REVIEW OF LITERATURE

Carl Hubel et al., (1995) in their study, concluded that triglycerides and free fatty acids, but not cholesterol, are increased in preeclampsia and correlate with the lipid preoxidation metabolite malondialdehyde[5]. These interactions contribute to endothelial cell dysfunction in preeclampsia.

Ray et al., (2006) studied the risk of preeclampsia in the presence of maternal triglyceridemia, another major element of the metabolic syndrome.[24] A total of 19 case control and 3 prospective cohort studies were included. In 14 studies, the mean TGL concentration was significantly higher among preeclamptic cases than among unaffected controls. In seven other studies, there was a nonsignificant trend in the same direction. The risk of preeclampsia typically doubled with each increasing TGL category. In the four studies, that adjusted for potential confounders, such as maternal age, parity and body mass index, there was a four fold higher risk of preeclampsia in the highest relative to the lowest triglyceride category.

Thus, the study concluded that there exists a consistent positive association between elevated maternal triglycerides and the risk of preeclampsia. Given that maternal hypertriglyceridemia is a common feature of the metabolic syndrome, interventional studies are needed to determine whether pre-pregnancy weight reduction and dietary modification can lower the risk of preeclampsia.

Sattar et al., (2002) studied the involvement of lipids in the pathophysiology of preeclampsia by doing lipid profile and lipoprotein subfactors in preeclampsia and in normal pregnancy.[25] They showed the potential links of the atherogenic lipid profile in preeclampsia to oxidative pathways, endothelial dysfunction and vascular inflammation.

Manten et al., (2004) studied the role of lipids in the endothelial cell dysfunction and thus leading to preeclampsia.[18] Seventy women were recruited, 50 were nulliparous women with singleton pregnancies and not in labour, 10 had severe preeclampsia. 20 had mild preeclampsia and 20 were healthy pregnant controls. The other 20 women were healthy non pregnant controls. There was a tendency to higher Lipoprotein-a concentration in women with preeclampsia. In the preeclampsia group, the concentrations of cholesterol and triglycerides were reported to be higher and the HDL cholesterol concentration was lower as compared to normal pregnancy.

Gratacos et al., (2003) evaluated the susceptibility to oxidation of LDL in women with a history of preeclampsia by a case control study.[9] Thirty five patients, who were diagnosed with severe preeclampsia and 35 controls were matched for age, BMI, smoking and parity. Plasma samples were analysed for total cholesterol, HDL, LDL, triglycerides and lipoprotein A. The invitro susceptibility to oxidation of LDL was measured and expressed in minutes.

Mean LDL cholesterol and triglyceride levels were higher in preeclampsia group compared with controls. The susceptibility to oxidation of LDL was also significantly

higher in the preeclampsia group.

Kaaja et al., (1995) and Sattar et al., (1997) have found higher plasma TGL and low HDL concentrations in women with preeclampsia and gestational HT.[12,26] Given these findings, it had been postulated that lipid abnormalities could play a role in the pathogenesis of preeclampsia, causing altered endothelial cell dysfunction and vascular damage.

Sattar et al., (1997) studied the lipoprotein subfraction concentrations in preeclampsia.[26] They concluded that the pathogenesis parallels to atherosclerosis.

In a recent study, in 2001, The National Heart Foundation of Australia found a correlation between F_2 - isoprotanes with increased levels of total and LDL cholesterol and triglycerides and significantly reduced levels of HDL cholesterol in women with preeclampsia, compared with that of the normotensive group. Increased levels of F_2 isoprotanes show that preeclamptic subjects are under increased oxidative stress, indicated by the increased rates of neutrophil activation.

Anceschi et al., (1992) conducted a study on erythrocyte membrane composition in pregnancy induced hypertension, which is an evidence for an altered lipid profile.[1] They concluded that the cholesterol / phospholipid ratio was significantly higher in the women with pregnancy induced hypertension compared with the normotensive pregnant women (PIH - 1.24 compared with normotensive - 0.88). The increased cholesterol /

phospholipid ratio of the erythrocyte membrane found in pregnancy induced hypertension represents one factor involved in the pathophysiology of this condition and a possible biochemical marker of the disease.

In a study by Dempsey et al., (2004), maternal birth weight was correlated negatively with triglycerides and correlated positively with high density lipoprotein cholesterol.[7] Women who weighed <2500 gms at birth had higher triglyceride and total cholesterol concentration and lower HDL concentrations, when compared with women who weighed 3000 to 3499 gms at birth. Women who were born small and became overweight in adulthood had less favourable lipid profiles than their counterparts who weighed >2500 gms at birth and remained lean.

Thus they concluded that factors that are related to growth in utero may help to predict the subsequent risk of altered lipid metabolism during pregnancy which may, in turn be causally related to the occurrence of preeclampsia.

In a study by Barden et al., (1999) the aim was to identify those factors in the non pregnant state that distinguished women who developed preeclampsia from those who had normotensive pregnancies.[2] The results were : Regardless of parity, women with preeclampsia had elevated BMI before, during and after pregnancy compared with women who had normotensive pregnancies. Triglycerides were significantly elevated in women who had preeclampsia both before and after delivery.

The relative elevation of blood pressure, BMI and lipids in the non pregnant state are features of the metabolic syndrome and may be important sensitising factors contributing to the pathogenesis of preeclampsia. A familial predisposition to preeclampsia may operate partly through these mechanisms.

Kokia et al., (1999) studied the maternal serum lipid profile in pregnancies complicated by hypertensive disorders.[15] Serum triglyceride levels were significantly elevated in the hypertensive patients. This elevation was not influenced by the severity or etiology of the hypertension. The lipid profile found in the hypertensive pregnant patients could be associated with enhancement of pathological lipid deposition in predisposed vessels such as the uterine spiral arteries. Further the hypertriglyceridemia found in the hypertensive patients may be associated with the hypercoagulability reported in pregnancy induced hypertension.

Daniel et al., (2004) investigated the relationship between early pregnancy plasma lipid concentration and risk of preeclampsia.[6] They concluded that early pregnancy dyslipidemia is associated with an increased risk of preeclampsia. This association may be significant in understanding the pathologic processes of preeclampsia and may help in developing strategies for prevention or early diagnosis of the disorder.

Thadhani et al., (1999) and Bodnar et al., (2005) found an association between high body mass index and hypercholesterolemia, which in turn increases the risks of preeclampsia.[28, 4]

Wolf et al., (2001) showed that obesity has a role to play in preeclampsia.[33] Ramsay et al., (2002) inferred that obesity is associated with dysregulation of metabolic, vascular and inflammatory pathways.[23] O'Brien et al., (2003) found that increased maternal BMI increases the risk of preeclampsia.[22]

Torun et al., (2001) found that dyslipidemia in early second trimester is mainly a feature of women with early onset pre-eclampsia.[29] Hubel et al., (1998) found elevated levels of low density lipoproteins and vascular cell adhesion molecule -1 in association with hyperlipidemia in preeclampsia.[11]

Ware Jauregui et al., (1999) compared the plasma lipid concentrations in preeclamptic and normotensive Peruvian women.[32] Vanderjagt et al., (2004) found that high - density lipoprotein and homocysteine levels correlated inversely in preeclamptic women.[30]

Kharba et al., (1998) determined the lipid peroxidation and vitamin E levels in preeclampsia.[14] Maseki et al., (1981) compared the lipid peroxide levels and lipid content of serum lipoprotein fractions of normotensive pregnant subjects and the subjects with preeclampsia.[19]

Gratacos et al., (1996) studied the variation in lipid levels during pregnancy in women with different types of hypertension.[10] Llurba et al., (2004) and Wakatsuki et al., (2000) did a comprehensive study of oxidative stress and the antioxidant status in

preeclampsia and normal pregnancy.[16,31] Granger et al., (2001) linked endothelial dysfunction with placental ischaemia, leading to the pathogenesis of preeclampsia.[8]

Sattar et al., (1997) studied the threshold effect of plasma triglyceride on appearance of small dense low density lipoproteins as well as the lipoprotein subfraction changes in normal pregnancy.[27] Lorentzen et al., (1995) found the fatty acid pattern of esterified and free fatty acids in sera of women with normal and preeclamptic pregnancy.[17]

Mikhail et al., (1995) showed the relationship between plasma triglyceride levels and severity of preeclampsia.[20] Khaliq et al., (2004) studied the serum lipid and lipoproteins in preeclampsia with special reference to parity.[13] Murai et al., (1997) correlated the maternal and fetal modulators of lipid metabolism with the development of preeclampsia.[21]

Aim

AIM

To analyse the lipid profile in normal pregnant women and the lipid profile changes in women with pregnancy induced hypertension.

Materials and Methods

MATERIALS AND METHODS

Study Design

Case Control Study.

Study period

September 2006 to April 2007.

Inclusion Criteria

1) Pregnant women in the third trimester diagnosed as PIH with no other associated complications.

2) Normal women in the third trimester of pregnancy with no other maternal medical complications, admitted for safe confinement.

Exclusion Criteria

- Chronic hypertension
- Pregestational Diabetes mellitus
- Nephrotic Syndrome
- Cardiac Disease.
- Hepatic Disease.
- Twin pregnancy
- Any medications except for vitamins and minerals.
- Smoking
- Ethanol use
- Labour contractions
- Thyrotoxicosis.

Methodology

Fasting blood samples were taken from 100 pregnant patients with Pregnancy Induced Hypertension (PIH) and 100 normal pregnant women, admitted in Kilpauk Medical College Hospital for safe confinement.

The samples were subjected to analysis of lipid profile.

Specimen

Freshly collected plasma. Anticoagulant used was EDTA.

Procedure

The lipid profile of the samples were determined using a semiautomated analyser.

Reagents used :

The following reagents were used for determination of the lipid values.

Total Cholesterol : Cholesterol Oxidase, Peroxidase

HDL Cholesterol : Phosphotungstate / Magnesium precipitation

LDL Cholesterol : Catalase / cholesterol esterase / cholesterol oxidase

Triglycerides : Glycerol Phosphate oxidase/ Peroxidase

Reference values of the lab

Total cholesterol	:	125 - 200 mgs / dl
HDL cholesterol	:	30 - 65 mgs / dl
Triglycerides	:	25 - 200 mgs / dl
LDL cholesterol	:	85 - 130 mgs / dl
VLDL	:	5 - 40 mgs / dl.

Results of the Study

RESULTS OF THE STUDY

- The women with pregnancy induced hypertension were grouped as Group I (Study Group).

- The normal pregnant women were grouped as Group II (Control Group).

The results of the study were analysed as follows

The results were analysed between group I and group II according to the age distribution, booked / unbooked status, obstetrics score, history of PIH in the previous pregnancy, body mass index, total cholesterol, HDL, VLDL, LDL, triglycerides, religion, dietary patterns, triglyceride levels in relation to the dietary pattern.

TABLE - 1

AGE

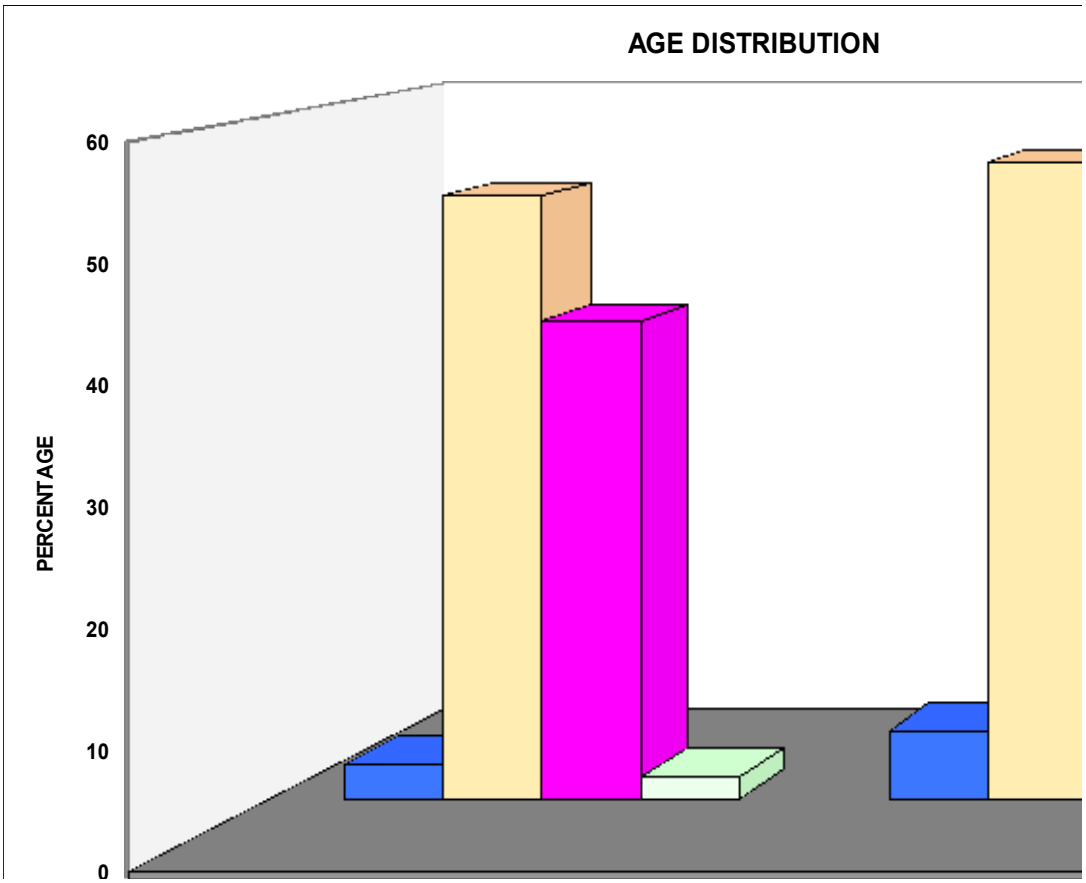
GROUPS	No. OF PATIENTS	MEAN AGE (Yrs)	S.D.	S.E. OF MEAN
GROUP I	100	25.1700	2.816	0.28817
GROUP II	100	24.6100	3.03147	0.30315

t = 1.330; p = 0.182 Not significant

TABLE - 1(a)

AGE DISTRIBUTION

AGE IN YEARS	GROUP I		GROUP II	
	NO. OF CASES	%	NO. OF CASES	%
< 20	3	3	6	6
21 - 25	53	53	56	56
26 - 30	42	42	34	34
31 - 35	2	2	4	4
TOTAL	**100**	**100**	**100**	**100**



Inference

There is no significant change in the age distribution between the two groups.

53% of the patients in Group I and 56% of patients in Group II were in the age group of 21 - 25 years.

Only 2% of patients in Group I and 4% of patients in Group II were in the age group of 31 - 35 years.

TABLE - 2

BOOKED / UNBOOKED

BOOKING STATUS	GROUP I		GROUP II	
	NO. OF CASES	%	NO. OF CASES	%
Booked	92	92	92	92
Unbooked	8	8	8	8
Total	**100**	**100**	**100**	**100**

$p = 100$; $x^2 = 0.000$ Not significant

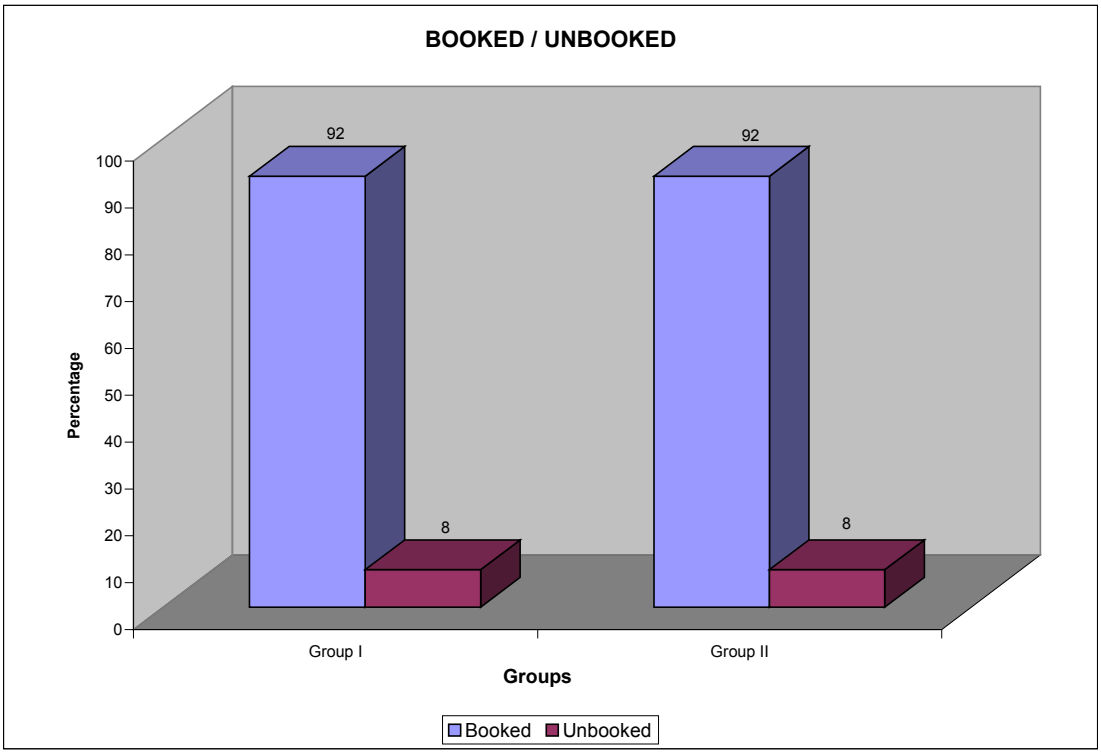


Fig. 2

Inference

The booking status of both the study group and the control group were the same, 92% being booked and 8% being unbooked.

TABLE - 3

OBSTETRIC SCORE

GRAVIDA	GROUP I		GROUP II	
	NO. OF CASES	%	NO. OF CASES	%
1	47	47	51	51
2	42	42	38	38
3	7	7	9	9
4	4	4	2	2
Total	**100**	**100**	**100**	**100**

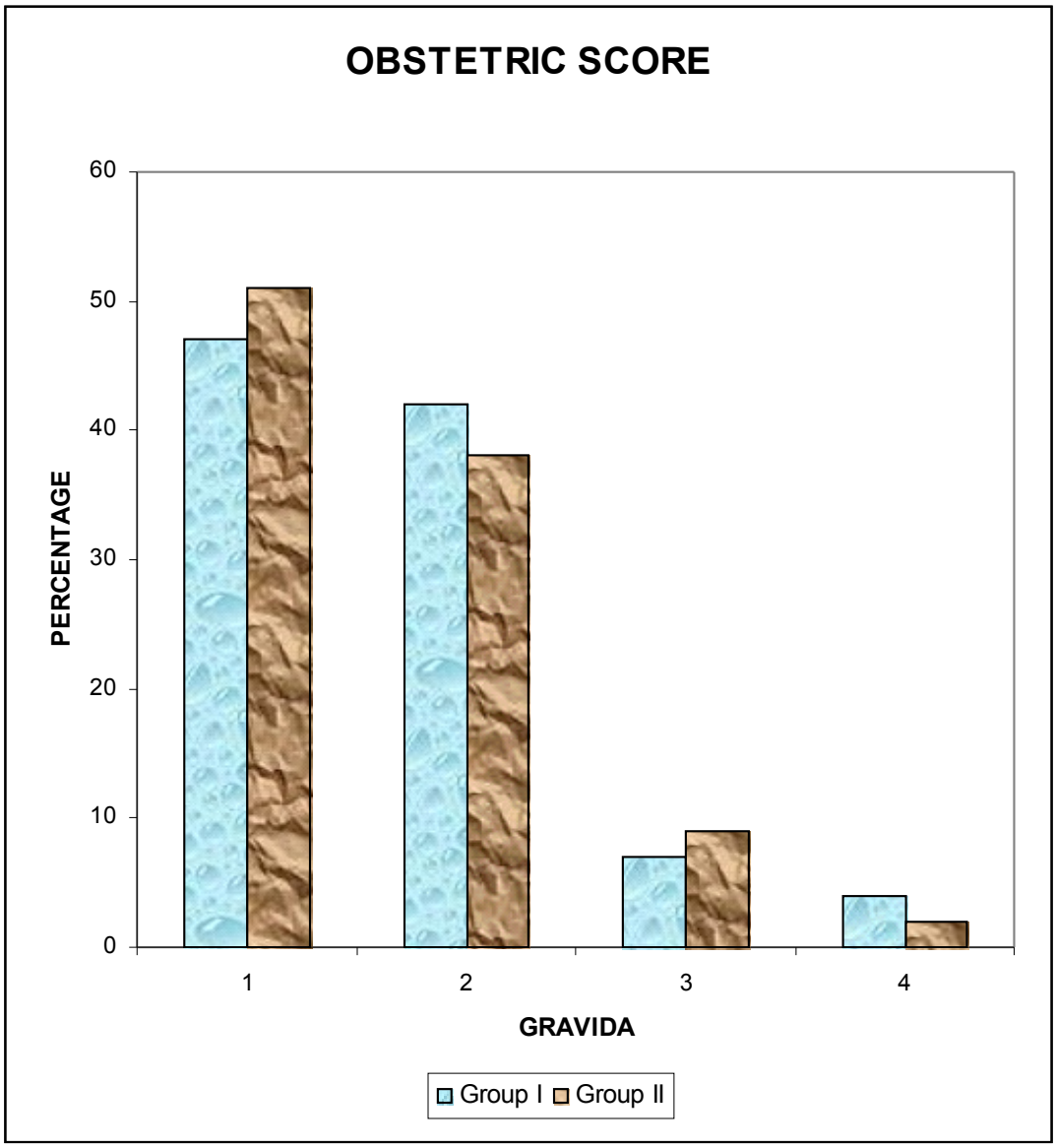


Fig. 3

Inference

47% of patients in Group I and 51% of patients in Group II were primigravida, while only 4% in Group I and 2% in Group II were Gravida 4.

TABLE - 4

HISTORY OF PIH IN PREVIOUS PREGNANCY

PIH IN PREVIOUS PREGNANCY	GROUP I		GROUP II	
	NO. OF CASES	%	NO. OF CASES	%
YES	12	12	5	5
NO	88	88	95	95
Total	**100**	**100**	**100**	**100**

$p = 0.076$; $x^2 = 3.150$ Not significant

Chi-square = 3.150

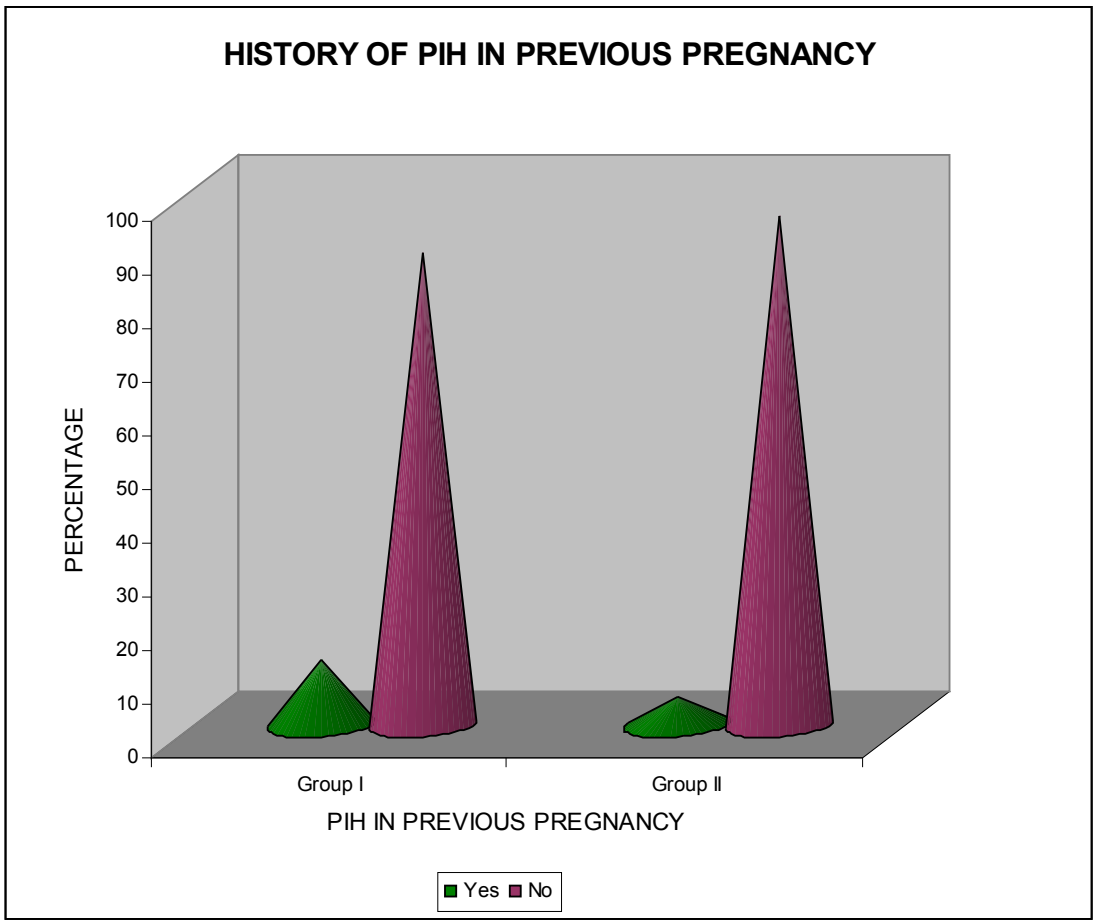


Fig. 4

Inference

12% of patients in group I and 5% of patients in Group II had H/o PIH in the previous pregnancy.

TABLE - 5

COMPARISON OF BMI BETWEEN THE TWO GROUPS

GROUP	NO. OF CASES	MEAN BMI	S.D.	S.E. OF MEAN
GROUP I	100	26.2080	3.39511	0.33951
GROUP II	100	24.710	2.52602	0.25260

p = 0.001 Significant

Inference

It is found that, patients with pregnancy induced hypertension (Group I) had high BMI than their normal counterparts (Group II)

TABLE - 6

TOTAL CHOLESTEROL LEVELS

GROUPS	NO. OF CASES	MEAN (mg%)	S.D.	S.E. OF MEAN
GROUP I	100	212.6420	53.10793	5.31079
GROUP II	100	173.7015	41.87940	4.18799

p = 0.000 Significant

Inference

The mean total cholesterol level is significantly higher in group I than in Group II.

TABLE - 7

HDL LEVELS

GROUPS	NO. OF CASES	MEAN (mg%)	S.D.	S.E. OF MEAN
GROUP I	100	48.0264	9.71615	0.97162
GROUP II	100	41.2684	9.35250	0.93525

p = 0.000 Significant

Inference

The mean HDL levels are higher in Group I compared to Group II.

TABLE - 8

VLDL Levels

GROUPS	NO. OF CASES	MEAN (mg %)	S.D.	S.E. OF MEAN
GROUP I	100	48.9432	18.38757	1.83876
GROUP II	100	35.5844	11.18140	1.11814

p = 0.000; T = 6.208 Significant

Inference

The mean VLDL values are significantly higher in Group I compared to Group II.

TABLE - 9

TGL Levels

GROUPS	NO. OF CASES	MEAN (mg %)	S.D.	S.E. OF MEAN
GROUP I	100	220.4673	62.97049	6.29705
GROUP II	100	169.2040	56.92760	5.69276

p = 0.000; t = 6.039 Significant

Inference

The mean triglyceride levels show a significant rise in Group I compared to that of

Group II.

TABLE - 10

LDL Levels

GROUPS	NO. OF CASES	MEAN (mg%)	S.D.	S.E. OF MEAN
GROUP I	100	135.2441	45.83614	4.58361
GROUP II	100	101.674	38.76126	3.87613

p = 0.000; T = 5.592 Significant

Inference

The mean LDL levels are also increased in Group I compared to that of Group II in a significant manner.

TABLE - 11

MODE OF DELIVERY

MODE OF DELIVERY	GROUP I		GROUP II	
	NO. OF CASES	%	NO. OF CASES	%
Vaginal	65	65	69	69
LSCS	35	35	31	31
TOTAL	**100**	**100**	**100**	**100**

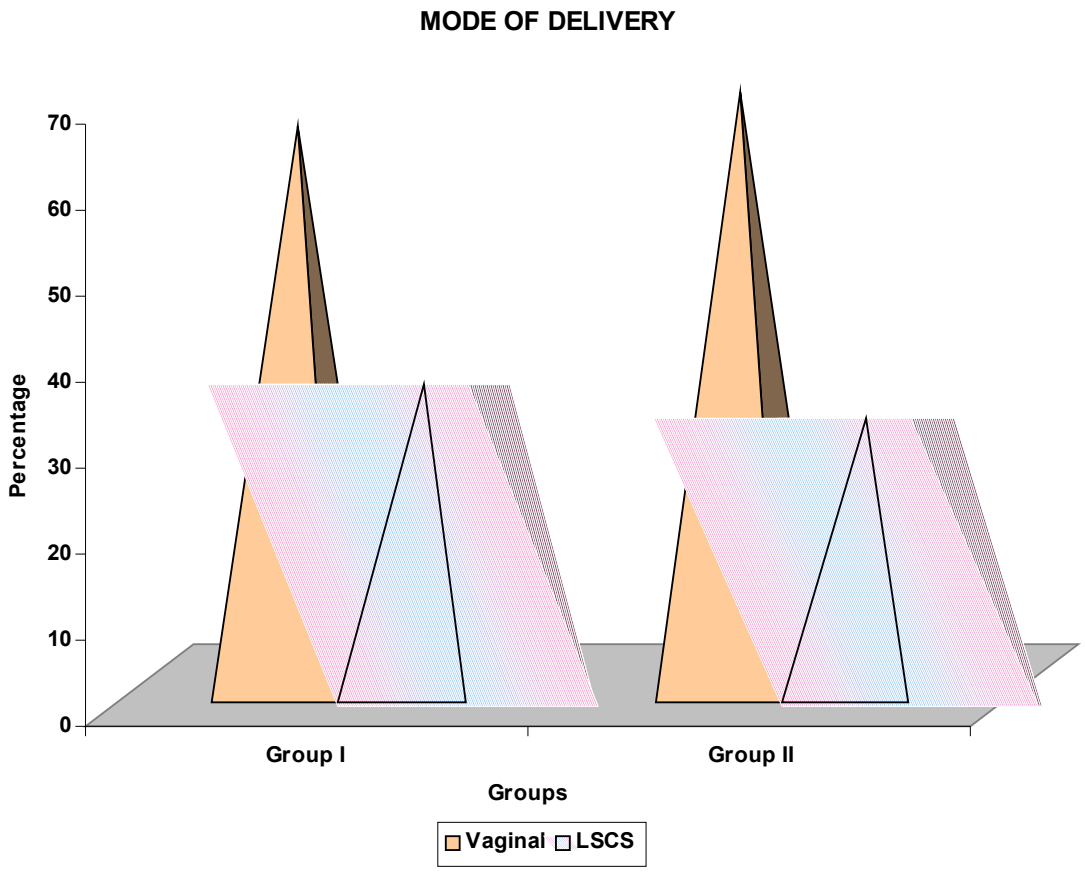


Fig. 5

Inference

65% of patients in Group I and 69% of patients in Group II had normal vaginal delivery

TABLE - 12

CORRELATION BETWEEN BMI & TGLS

GROUP I

BMI	NO. OF CASES	MEAN OF TGLS (mg%)	S.D.	S.E. OF MEAN
Normal	62	199.3725	53.74081	6.82509
High / OBESE	37	254.0874	63.4071	10.42795

p = 0.000 Significant

GROUP II

BMI	NO. OF CASES	MEAN OF TRIGLYCERIDES (mg %)	S.D.	S.E. OF MEAN
Normal	82	161.6781	51.8977	5.80234
High / OBESE	18	203.5611	69.93061	16.01140

$p = 0.000$; $t = 2.915$ Significant

Inference :

- It is found that the obese patients or the patients with high BMI in Group I had elevated triglycerides than those with normal BMI in the same group.

- Similarly, the patients with high BMI / obese patients in Group II also had elevated triglycerides than the normal.

TABLE - 13

NON OBESE GROUP

GROUPS	NON OBESE	MEAN TGL (mg %)	S.D.	S.E. OF MEAN
GROUP I	62	127.8315	44.97383	5.71168
GROUP II	81	96.9215	36.13983	4.01554

p = 0.000; t = 4.557 Significant

TABLE - 13 (a)

OBESE GROUP

GROUPS	OBESE	MEAN TGL (mg %)	S.D.	S.E. OF MEAN
GROUP I	37	254.0874	63.43071	10.42795
GROUP II	18	203.5611	67.93061	16.01140

Variance - 25 - F = 0.676;

Significant = 0.415

2 tailed = 0.009

p = 0.009

Inference :

Among the non obese group, the PIH patients in Group I had increased triglycerides compared to the normal women in Group II.

Among the obese group also, the mean triglyceride values were higher in patients with pregnancy induced hypertension compared to the normotensive pregnant women.

TABLE - 14

CLASSIFICATION OF THE GROUPS ACCORDING TO RELIGION

GROUPS	RELIGION					
	HINDUS		MUSLIMS		CHRISTIANS	
	NO. OF PATIENTS	%	NO. OF PATIENTS	%	NO. OF PATIENTS	%
GROUP I	73	73	15	15	12	12
GROUP II	88	88	4	4	8	8

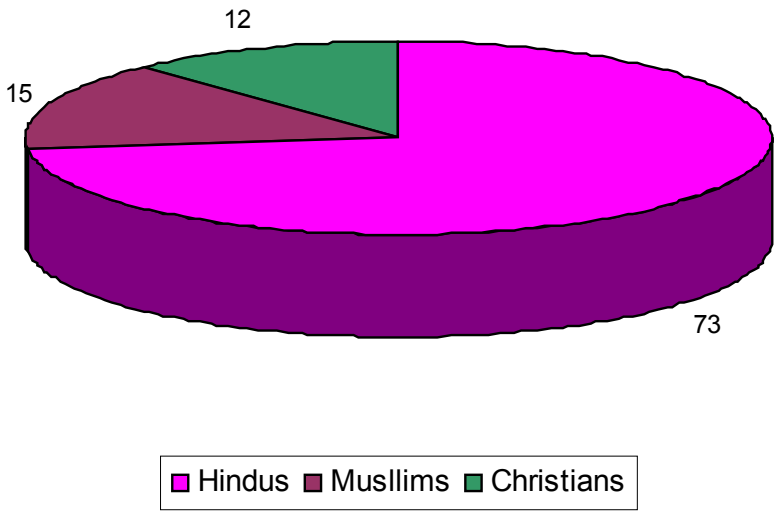
Inference

Among the population in Group I, 73% of the women were Hindus, 15% were Muslims and 12% were Christians.

Among the population in Group II, 88% of the women were Hindus, 4% were Muslims and 8% were Christians.

CLASSIFICATION OF THE GROUPS ACCORDING TO RELIGION

GROUP I



GROUP II

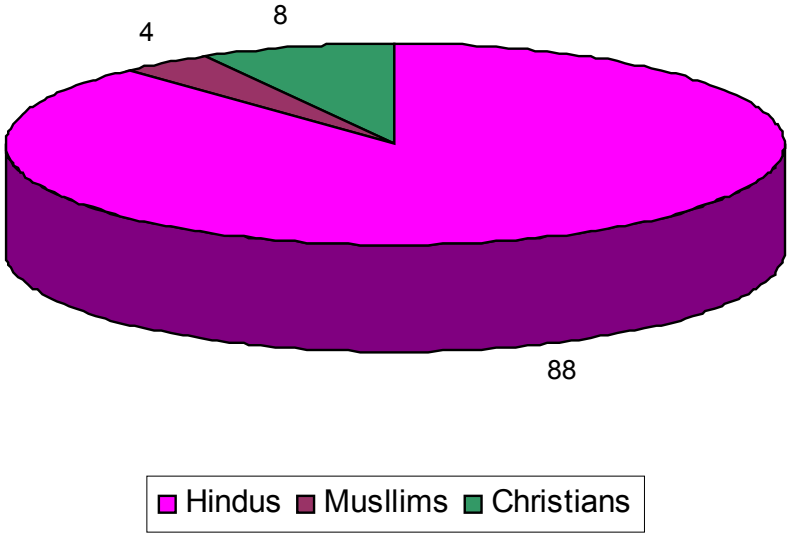


Fig. 6

TABLE - 15

DIETARY PATTERN

GROUPS	MORE THAN 2 SERVES OF NON-VEG. DIET PER WEEK		
	HINDUS (%)	MUSLIMS (%)	CHRISTIANS (%)
GROUP I	30	80	66
GROUP II	40	100	63

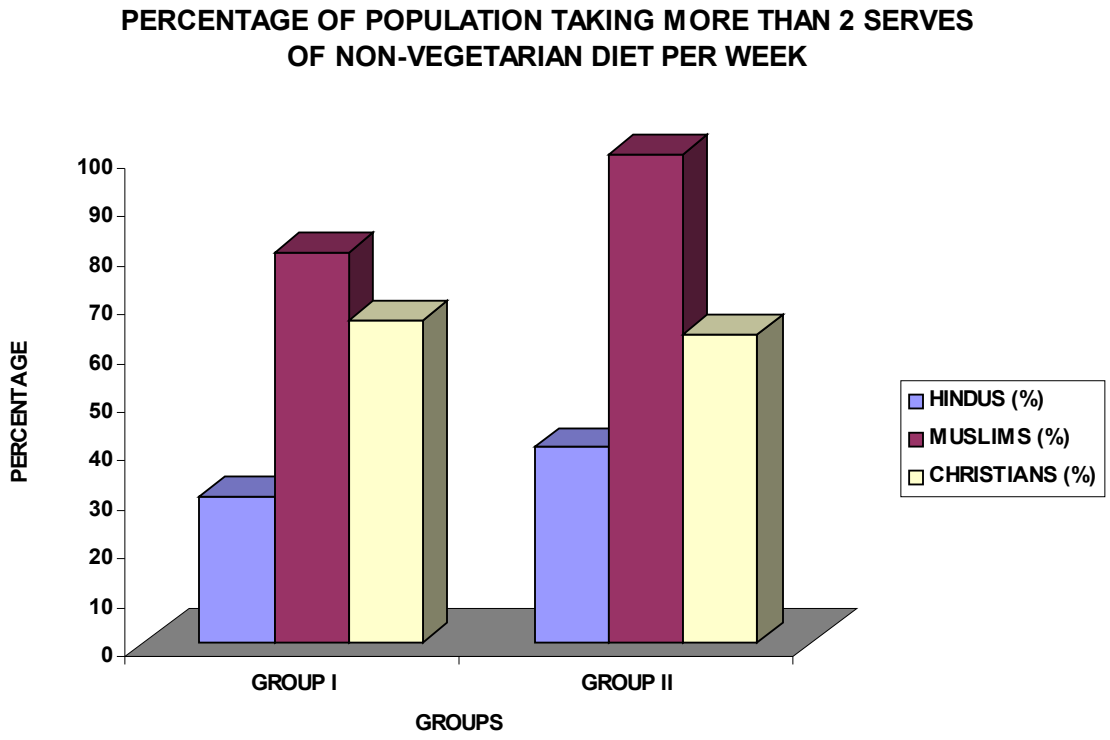


Fig. 7

Inference

Among the population in Group I, 80% of Muslims, 66% of Christians and 30% of Hindus took more than two serves of non-vegetarian diet per week.

In group II, 100% of Muslims, 63% of Christians and 40% of the Hindus took more than two serves of non-vegetarian diet per week.

TABLE – 16

CORRELATION OF ELEVATED TGLS WITH RELIGION

GROUPS	RELIGION								
	HINDUS			MUSLIMS			CHRISTIANS		
	↑TGL	NORMAL TGL	% ↑TGL	↑TGL	NORMAL TGL	% ↑TGL	↑TGL	NORMAL TGL	% ↑TGL
Group I	35	38	40	11	4	73	7	5	58
Group II	19	69	21.5	1	3	25	1	67	12.5

CORRELATION OF ELEVATED TGLs WITH RELIGION

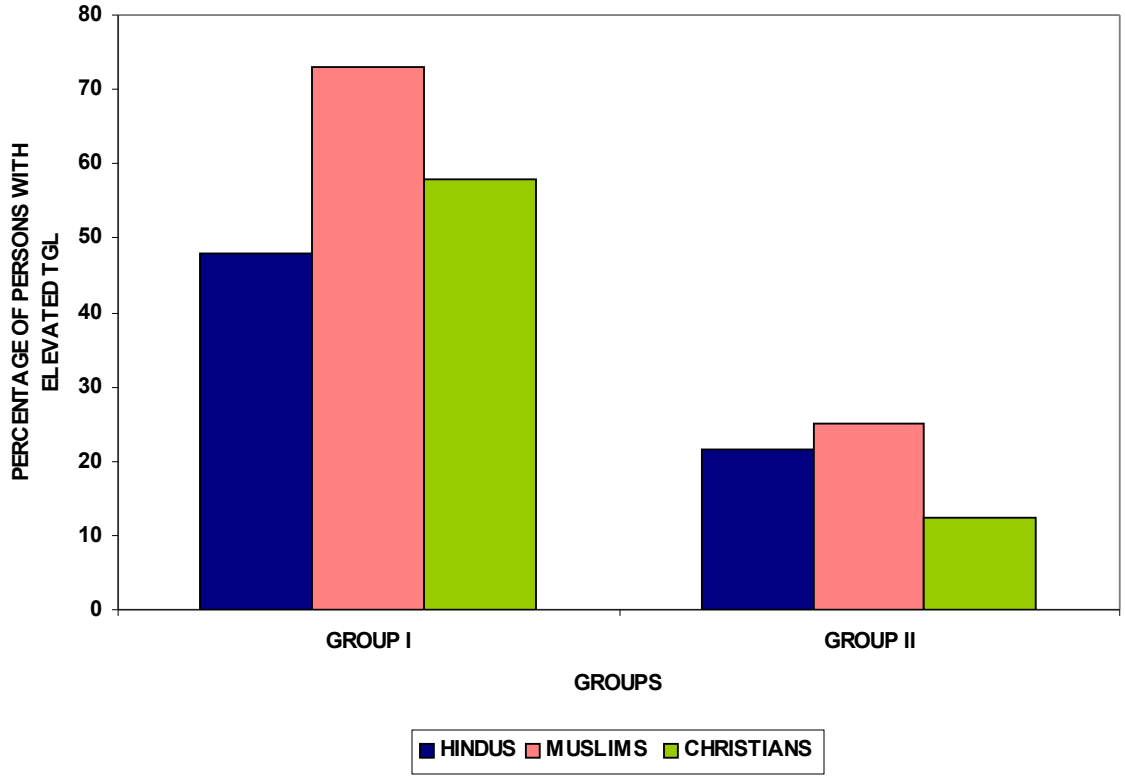


Fig. 8

Inference

Among the population in Group I, 73% of Muslims, 48% of Hindus and 58% of Christians had elevated triglyceride levels.

Among the population in Group II, 25% Muslims, 12.5% of Christians and 21.5% of Hindus had elevated triglyceride levels.

TABLE - 17

CORRELATION BETWEEN DIETARY PATTERN AND TRIGLYCERIDE LEVELS

GROUP - I

TRIGLYCERIDE PATTERN	PERSONS TAKING MORE THAN 2 SERVES OF NON - VEG. PER WEEK			TOTAL
	HINDUS	MUSLIMS	CHRISTIANS	
Elevated	19	11	7	37
Normal	3	1	1	5

Inference

Among Group I, 88% of persons taking more than two serves of non-vegetarian diet had elevated triglyceride levels.

TABLE - 18

CORRELATION BETWEEN DIETARY PATTERN AND TRIGLYCERIDE LEVELS

GROUP - II

TRIGLYCERIDE PATTERN	PERSONS TAKING MORE THAN 2 SERVES OF NON - VEG. PER WEEK			TOTAL
	HINDUS	MUSLIMS	CHRISTIANS	
Elevated	12	1	1	14
Normal	23	3	4	30

Inference

Among Group II, 27.2% of persons taking more than 2 serves of non-vegetarian diet per week had elevated triglyceride levels.

Discussion

DISCUSSION

In our study, it is found that there is no significant change in age distribution between the two groups.

Our study , similar to that by Ray et al., (2006) shows that the mean triglyceride concentration was significantly higher among the preeclamptic cases than among the unaffected controls.[24] What might hypertriglyceridemia predispose a woman to preeclampsia, if there truly exists a causal relationship? A likely factor is the higher risk of placental vasculopathy. Persons with the metabolic syndrome of which hypertriglyceridemia is a major feature, display evidence of chronic inflammation, hypercoagulability and endothelial dysfunction.

Our study similar to Carl Hubel et al., (1995) shows that triglycerides and free fatty acids are increased in preeclampsia.[5] Their study correlated the interaction of the lipid peroxidation metabolite, malondialdehyde to endothelial cell dysfunction in preeclampsia. However the effects of malondialdehyde are not included in our study.

The study by Ray et al., (2006) concluded that there exists a correlation between obesity and the risk of preeclampsia.[24] Our study also shows that women with elevated BMI in both the control and the PIH groups had elevated levels of triglycerides and LDL cholesterol. The current study also shows hypertriglyceridemia associated with maternal obesity. Maternal obesity, diabetes mellitus and chronic hypertension, the major features

of the metabolic syndrome are positively correlated with the development of preeclampsia in many studies.

The study by Barden, et al., (1999) shows that regardless of parity women with preeclampsia had elevated BMI during pregnancy compared with women who had normotensive pregnancies and triglycerides were significantly elevated in these women and the levels of triglycerides after 6 weeks of delivery were found to the decreased. [2] However in our study, the levels of postpartum triglycerides were not measured.

Our study similar to Sattar et al., (1997) shows the elevated levels of cholesterol and trigycerides.[26] Kaaja et al., (1995) has found high plasma triglycerides and low HDL concentrations in women with preeclampsia and gestational hypertension.[12] In our study though there is elevated level of cholesterol, and triglycerides, the HDL level is found to be normal.

This study shows that lipid abnormalities can lead to endothelial cell dysfunction and vascular damage, thus playing a role in the pathogenesis of preeclampsia.

The National Heart Foundation of Australia found increased levels of F_2 Isoprotane in patients with increased LDL and triglyceride concentrations. Further studies are required in finding the association of F_2 Isoprotanes with preeclampsia. They also found significantly reduced HDL levels in preeclamptic patients while in our study,

the levels of HDL were normal. Further more studies are thus required in determining the association between HDL levels and predisposition to preeclampsia.

Bendomir et al., (1997) studied that lipoprotein subfraction concentrations are higher in preeclampsia.[3] In our study, the lipoprotein subfractions were not studied. Further studies are required to find the effect of lipoprotein subfractions in pregnancy.

Anceschi et al., (1992) found increased cholesterol / phospholipid ratio of the erythrocyte membrane in women with pregnancy induced hypertension.[1] In our study, the levels of the membrane phospholipids were not measured.

Dempsey et al., (2004) found that women who were small at birth and became overweight in adulthood had an unfavourable lipid profile.[7] But the subjects in our study were not aware of their birth weight. Hence this association factor could not be correlated in our study.

Our study similar to that by Kokia et al., (1999) shows significantly elevated levels of triglycerides in the women with pregnancy induced hypertension.[15]

Furthermore, in our study we have found a correlation between the dietary pattern and the levels of triglycerides. It is found that 80% of the Muslim population in the PIH group took more than two serves of non - vegetarian diet per week and among them 73% of the Muslims had elevated triglyceride levels.

In the Hindu population, 30% of PIH group took more than two serves of non-vegetarian diet per week and among them 48% had elevated triglyceride levels.

In Group I patients, 30% of Christians took more of non-vegetarian diet and 12.5% of them had elevated triglyceride levels.

The proportion of persons taking more of non-vegetarian diet was comparatively lesser in Group II and a lower proportion of them had elevated triglyceride levels.

Thus there exists a causal association between the dietary pattern and the lipid profile. Increased servings of non-vegetarian diet had an adverse effect on lipid profile with increased triglyceride levels. This in turn places the individuals to be in high risk category towards the development of preeclampsia. Thus a dietary modification in early pregnancy and perhaps pre pregnancy state too may help in the prevention of progress of the atherosis process in the placenta. Further studies are thus required in this background of dietary factor. Future research could be done in the modification of dietary pattern by advocating more of fibre diet and less of fatty diet and finding whether this helps in the prevention of preeclampsia to a certain extent.

Thus, the adverse lipid profile found in the hypertensive pregnant patients could be associated with the enhancement of pathological lipid deposition in the predisposed vessels such as uterine arteries.

COMPARISON OF THE LIPID PROFILE ANALYSIS IN VARIOUS STUDIES

	TGLS	**LDL**	**HDL**
Ray et al.	↑	-	-
Carl Hubel et al	↑	-	-
Sattar et al	↑	↑	-
Kaaja et al	↑	↑	↓
Our study	↑	↑	normal

Summary

SUMMARY

(1) There is no significant change in the age distribution between the two groups. 53% of the patients in Group I and 56% of patients in Group II were in the age group of 21 - 25 years. Only 2% of patients in Group I and 4% of patients in Group II were in the age group of 31 - 35 years.

(2) The booking status of both the study group and the control group were the same, 92% being booked and 8% being unbooked.

(3) 47% of patients in Group I and 51% of patients in Group II were primigravida, while only 4% in Group I and 2% in Group II were Gravida 4.

(4) 12% of patients in group I and 5% of patients in Group II had H/o PIH in the previous pregnancy.

(5) It is found that, patients with pregnancy induced hypertension (Group I) had high BMI than their normal counterparts (Group II).

(6) The mean total cholesterol level is higher in group I than in Group II.

(7) The mean HDL levels are higher in Group I compared to Group II.

(8) The mean VLDL values are higher in Group I compared to Group II.

(9) The mean triglyceride levels show a significant rise in Group I compared to that of Group II.

(10) The mean LDL levels are also increased in Group I compared to that of Group II.

(11) 65% of patients in Group I and 69% of patients in Group II had normal vaginal delivery.

(12) It is found that the obese patients or the patients with high BMI in Group I had elevated triglycerides than those with normal BMI in the same group.

Similarly, the patients with high BMI / Obese patients in Group II also had elevated triglycerides than the normal, in Group II.

(13) Among the non obese group, the PIH patients in Group I had increased triglycerides compared to the normal women in Group II.

Among the obese group also, the mean triglyceride values were higher in patients with pregnancy induced hypertension compared to the normotensive pregnant women.

(14) Among the population in Group I, 73% of the women were Hindus, 15% were Muslims and 12% were Christians.

Among the population in Group II, 88% of the women were Hindus, 4% were Muslims and 8% were Christians.

(15) Among the population in Group I, 80% of Muslims, 66%, of Christians and 30% of

Hindus took more than two serves of non-vegetarian diet per week.

In group II, 100% of Muslims, 63% of Christians and 40% of the Hindus took more than two serves of non-vegetarian diet per week.

(16)Among the population in Group I, 73% of Muslims, 48% of Hindus and 58% of Christians had elevated triglyceride levels. Among the population in Group II, 25% Muslims, 12.5% of Christians and 21.5% of Hindus had elevated triglyceride levels.

(17)Among Group I, 88% of persons taking more than two serves of non-vegetarian diet had elevated triglyceride levels.

(18)Among Group II, 27.2% of persons taking more than 2 serves of non-vegetarian diet per week had elevated triglyceride levels.

Conclusion

CONCLUSION

Analysing and comparing the results between the study group and the control group, it was concluded that :

- 53% of the study group were in the age group of 21 - 25 years.
- 47% of the study group were primigravida.
- BMI was elevated in the PIH group.
- Triglycerides and LDL were elevated in the PIH group, compared with the control group.
- The obese group with elevated BMI had elevated triglyceride levels.

Thus there exists a consistent positive association between elevated maternal triglyceride and the risk of preeclampsia. Given that the maternal hypertriglyceridemia is a common feature of the metabolic syndrome, interventional studies are needed to determine whether pre -pregnancy weight reduction and dietary modification can lower the risk of preeclampsia.

The matter of whether triglycerides share a causative relationship with preeclampsia should be expanded to the study of other lipoprotein particles and microparticles, as well as a detailed analysis of the microvascular bed of the delivered

placenta. The collection of blood specimens in early pregnancy measuring concentrations of insulin, glucose and inflammatory markers, alongside anthropometrics assessment and then followed by a thorough assessment of clinical outcomes through a large cohort study might optimally address the role of triglycerides and the metabolic syndrome in the causation of preeclampsia.

Clearly, there is a need to establish whether preconception dietary modification, such as adoption of a Mediterranean diet among obese women, can reduce the future risk of preeclampsia and other placenta mediated diseases, including placental abruption and foetal intrauterine growth restriction.

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Master Chart

KEY TO MASTER CHART

S.No.	-	Serial Number
IP NO.	-	Inpatient Number
B	-	Booked
UB	-	Unbooked
POA Wks	-	Period of amenorrhoea in weeks
SEC	-	Socio Economic Status
PIH in PP	-	Pregnancy induced hypertension in past pregnancy.
BMI	-	Body Mass Index
BP	-	Blood Pressure
TC	-	Total Cholesterol
HDL	-	High Density Lipoproteins
LDL	-	Low density Lipoproteins
VLDL	-	Very low density lipoproteins
TGL	-	Triglycerides
MOD	-	Mode of delivery
R	-	Religion
Diet	-	>2 servings of non vegetarian diet per week
CS	-	Caesarean Section
V	-	Vaginal delivery
C	-	Christian
H	-	Hindu
M	-	Muslim
G	-	Gravida
P	-	Para
L	-	Live Children

PROFORMA

Name :

Age :

IP No. :

Obstetric Score :

Booking Status :

Socio Economic Status :

H/o PIH in Previous Pregnancy :

Diet History : Vegetarian

-

Non Vegetarian

- Taking more than two serves per week
- Taking less than two serves per week.

Family History :

Religion :

Chief Complaints :

H/o Swelling of legs / oliguria

Any H/o Headache / Blurring of vision / Vomiting / Epigastric pain.

Any H/o DM/HT/Bronchial Asthma /Cardiac/Thyroid Disease.

General Examination

Anaemia Jaundice Pedal edema JVP

Vitals:

PR: BP: RR:

Any symptoms of imminent eclampsia.

Systemic examination:

Respiratory system.

Cardiovascular System.

CNS:

Obstetric Examination

Investigations:

Urine
- Albumin
- Sugar
- Deposits

24 hrs urinary proteins

VDRL

NVP

Blood grouping & typing

CBC - Platelet Count

Bleeding time

Clotting time

Blood Sugar

Urea

Sr. Creatinine

Sr.Uric acid.

Sr.Fibrinogen

Liver function Tests

Lipid Profile

Fundus Examination

Obstetric Ultrasound.

LIST OF ABBREVIATIONS USED

BMI	-	Body Mass Index
dl	-	decilitre
Fig.	-	Figure
H/o	-	History of
HDL	-	High Density Lipoproteins
HT	-	Hypertension
LDL	-	Low Density Lipoproteins
LSCS	-	Lower Segment Caesarean Section
mgs	-	milligrams
PIH	-	Pregnancy Induced Hypertension
S.D.	-	Standard Deviation
S.E.	-	Standard Error
TGLS	-	Triglycerides

VLDL	-	Very Low Density Lipoproteins
yrs	-	Years
%	-	Percentage